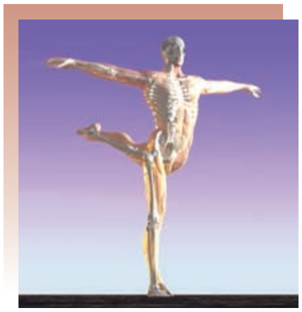

Simplifying the Complexities of Maintaining Balance

Insights Provided by Simple Closed-Loop Models of Human Postural Control

ROBERT J. PETERKA

How do we maintain our balance? This might seem to be a trivial problem since most of us are able to maintain our balance in a variety of environments without apparent effort. However, further consideration reveals that this problem is not trivial since complex biomechanical, sensory, neural, and muscular subsystems are involved in human postural control. Each one of these subsystems is so dauntingly complex that one might be discouraged to think that any system involving all of these subsystems could be understood. In developing a quantitative model to help us understand the postural control system, one might be tempted to capture as much of the complexity as is known about each of the subsystems. However, this article will follow the approach of Occam's Razor [1]. That is, we begin with the simplest possible representation of each of the subsystems and only add complexity as necessary to be consistent with experimental data.

Simplest Feedback Control Model

As a first step in model development, simplify the biomechanics of the body. The biomechanics of a multilink inverted pendulum are well known but quite complex [2]. However, when the body is represented as a single-link inverted pendulum with body rotation occurring about the ankle joint axis, the equations of motion are quite simple. When a subject's body-in-space angular orientation (*BS*) deviates from upright, gravity (*g*) acts on the center-of-mass of the body (COM; body mass *m* located at a height *h* above the ankle joint axis) producing a destabilizing torque equal to *mgh* sin(*BS*), which in turn produces a rotational acceleration away from the upright position that is inversely proportional to the moment-of-inertia (*J*) of the body about the ankle joint axis. To maintain balance, a corrective torque (T_c) must be applied to counter the torque due to gravity. The differential equation of an inverted pendulum is given by:

$$J\frac{d^2BS}{dt^2} = mgh\sin(BS) + T_c. \quad (1)$$

After making the small angle approximation *BS*≈sin(*BS*) and taking the Laplace transform, (1) can be expressed as a transfer function relating *BS* to T_c :

$$\frac{BS(s)}{T_c(s)} = \frac{1}{Js^2 - mgh} \quad (2)$$

where *s* is the Laplace transform variable.

As a second step, simplify the sensory systems involved in postural control. It is well known that sensory orientation visual, vestibular, and proprioceptive/somatosensatory orientation cues contribute to postural control since stimulation of these sensory systems evokes body sway [3]. To simplify, eliminate vision by eye closure and consider the postural responses of a subject with absent vestibular function. This leaves only proprioception/somatosensation, which can be further simplified by including only proprioception, which we define narrowly as a sensory cue signaling body tilt relative to the support surface (*SS*) upon which the subject stands. Finally, proprioception is assumed to have no dynamics and is represented by a weighting factor (W_{prop}), which can be set equal to 1 without loss of generality.

As a third step, assume that our simplified postural control system is configured as a position feedback control system [Figure 1(a)]. Feedback is accomplished by the mechanical relationship *BF*=*BS*–*FS*. That is, body orientation relative to the feet (*BF*) is the difference between body orientation relative to earth-vertical (body-in-space, *BS*) and foot-in-space orientation (*FS*). *BF* is sensed by proprioceptors to form an internal position error signal (*e*), which in turn is used by neuromuscular systems to generate T_c. Finally, T_c produces a change in *BS* according to (2). A time delay is included in the control system that represents the combined delays due to sensory reception, neural transmission, neural processing, muscle activation, and force development.

As a fourth step, make some initial assumptions about the "neural controller" that represents the neuromuscular processing that leads to corrective torque generation. It is known that the minimal requirement for stabilization of an inverted pendulum body is that corrective torque is generated in proportion to both *e* (with a "stiffness" proportionality factor K_P), and the time derivative of *e* (with a "damping" factor K_D) [4]. That is, the neural controller includes two components that provide "PD" control. By analogy to man-made control systems, we might also consider a third component proportional

0739-5175/03/$17.00©2003IEEE

Consider the possibility that sensory cues from proprioception/ somatosensation contribute more than just a position-related signal for postural control but also a force-related signal.

to the integral of e (proportionality factor K_I) so that the overall controller provides "PID" control [5].

As a fifth step, form the overall transfer function of this feedback control system. This transfer function is given by:

$$\frac{BS(s)}{FS(s)} = \frac{W_{\mathrm{prop}} \cdot NC \cdot B \cdot TD}{1 + W_{\mathrm{prop}} \cdot NC \cdot B \cdot TD} \qquad (3)$$

where B is the inverted pendulum body transfer function given by (2), NC is the neural control transfer function given by K_P+K_Ds for PD control or K_P+K_Ds +K_I/s for PID control, and TD is the time delay given by $e^{-\tau_d s}$.

Finally, compare experimental results with model predictions to determine if the above simplifications are justified or if additional factors must be included to account for the experimental data. All experimental data were collected from subjects who gave their informed consent to perform protocols approved by the IRB of Oregon Health & Science University. The four panels in Figure 2 show different transfer function fits to identical sets of experimentally derived transfer function gain and phase data points. The experimental data were from one test trial of a bilateral vestibular loss subject standing eyes closed on a support surface that tilted about the ankle joint axis according to a pseudorandom stimulus profile with a 1° peak-to-peak amplitude [6]. Single-link inverted pendulum biomechanics were assured by use of a backboard assembly that permitted only anterior-posterior body sway about the ankle joint axis. Transfer function gain and phase measures as a function of stimulus frequency were computed from power and cross power spectra of the FS and BS waveforms [7]. The gain represents the ratio of BS to FS, and it is assumed that the feet remained in contact with the support surface so that $FS = SS$. A gain of 1 and phase of 0° indicates that the subject's body was perfectly oriented to the support surface, while a gain of zero indicates that the body remained oriented to earth-vertical independent of the surface orientation.

Experimental results showed that gains were largest at stimulus frequencies in the 0.2-0.5 Hz range and decreased at lower and higher frequencies. Phase was close to 0° at about 0.15 Hz. A phase lead was present at lower frequencies, and an increasing phase lag developed with

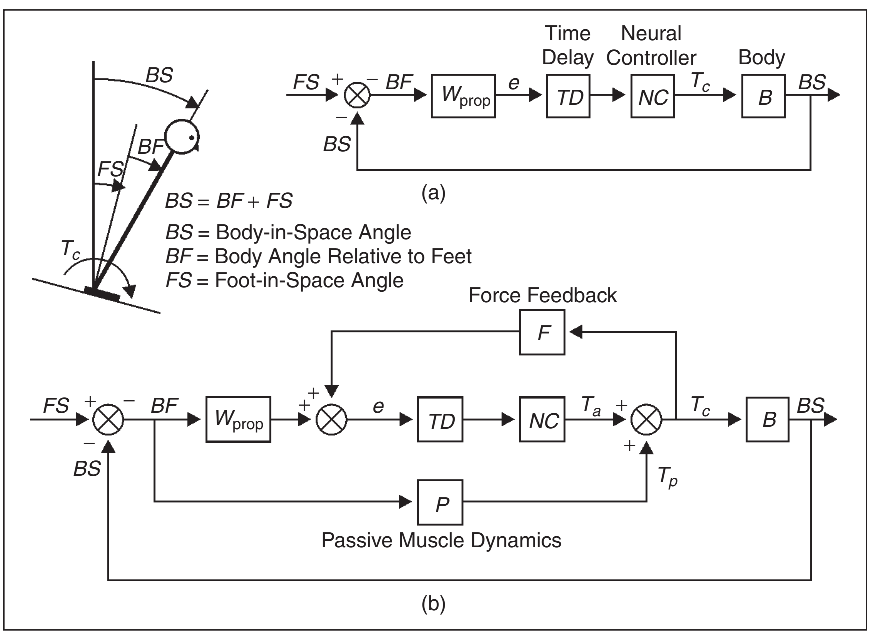

Fig. 1. (a) A simple postural control model formulated as a negative feedback position control system with sensory orientation information from proprioceptors sensing body orientation relative to the support surface. A weighting factor W_{prop} is applied to proprioceptive sensory information, and this information is processed by a neural controller to generate corrective torque T_c. The stick figure defines the positive directions of orientation angles and corrective torque. (b) Modified model that includes passive muscle dynamics P and a positive feedback loop conveying force-related sensory information F. Passive muscle properties generate passive corrective torque T_p that sums with active torque T_a derived from sensory information to give the overall corrective torque T_c applied to the body.

Weight changes must be performed in a coordinated fashion since changing the internal gains of a feedback control system can have a profound effect on the dynamics of the system.

increasing frequency consistent with the presence of a time delay in the control system.

The question is whether or not the simple model given by (3) is capable of accounting for these experimental results. Figure 2(a) shows a curve fit (solid lines) of (3) with a PD neural controller. In this fit, the neural controller parameters K_P and K_D and the time delay τ_d were allowed to vary. Body parameters m, h, and J were measured or estimated using anthropometric methods [8]. This model fit did not provide a good explanation of the experimental data. In particular, this model was incapable of accounting for the experimentally observed decline in low-frequency gain and the low-frequency phase lead.

A considerably better fit to the experimental data was obtained if the neural controller had PID characteristics [Figure 2(b)]. Mid-frequency data (0.1-1 Hz) were quite well described by this model. For frequencies less than 0.1 Hz, the model with PID control improved the fit by accounting for a gain decline with decreasing frequency and some phase lead. However, the model predicted that the phase will approach 0° at low frequencies, while the experimental data showed no sign of phase returning to 0°. Additionally, for frequencies above about 1 Hz, the experimental data showed more phase lag than predicted by the fit. These discrepancies indicate that some additional factors not considered in this simple model are contributing to the dynamic behavior of the postural control system.

Modified Postural Control Model

The close correspondence between the model and data in Figure 2(b) suggests that we are on the right track but are possibly missing some important component(s). Consider the possibility that sensory cues from proprioception/somatosensation contribute more than just a position-related signal for postural

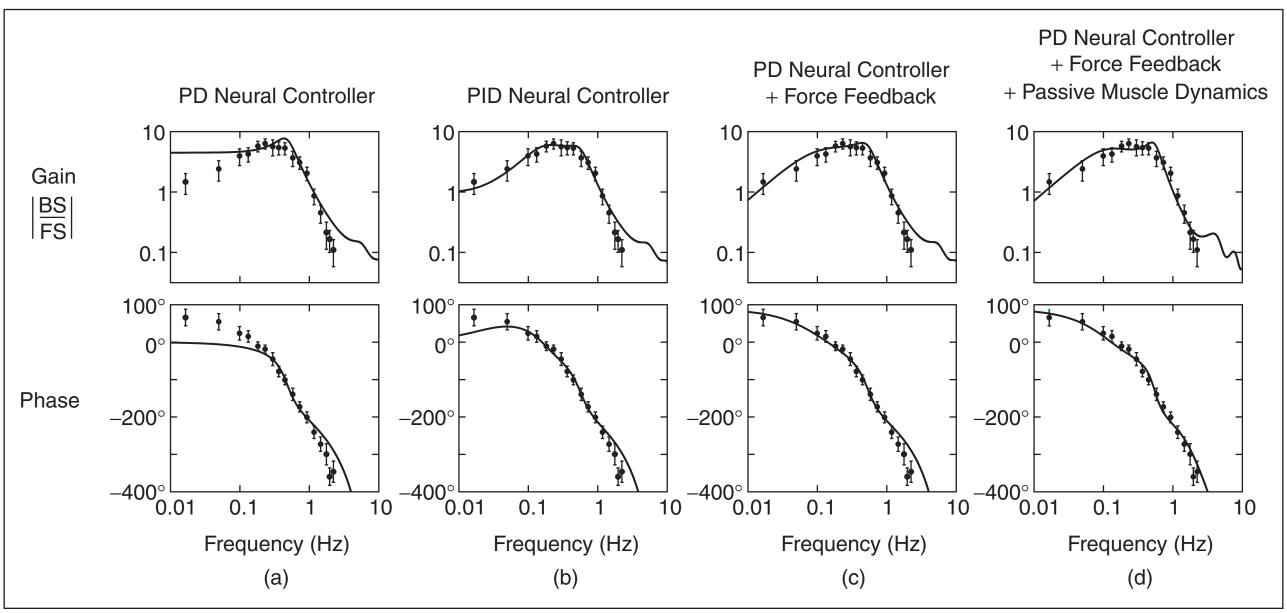

Fig. 2. Curve fits of model equations (solid lines) to experimental transfer function data (points showing mean with 95% confidence limits) obtained from a bilateral vestibular loss subject standing with eyes closed. Posture was perturbed by a pseudorandom rotation of the support surface (1° peak-to-peak amplitude) about an axis aligned with the ankle joint axis. Panels (a)-(d) show different model fits to the same set of experimental gain and phase data.

control but also a force-related signal. A force-related signal might arise from the pressure distribution on the feet [9] or muscle tension signaled by Golgi tendon organs [10]. The model shown in Figure 1(b) postulates that force-related sensory information is used in a positive feedback loop that sums with position-related information obtained by negative feedback to form an internal error signal e. Then T_c is generated through the action of a PD controller. To understand how positive force feedback can contribute to postural control, consider a subject who is leaning backward. A positive torque T_c is necessary to hold this position and prevent a backward fall. Pressure on the heels of the feet or tension in muscles on the front of the legs due to this backward lean is sensed and contributes to an increase in the internal error e, which results in a further increase in T_c, causing a return of body orientation toward the upright position. Thus, positive feedback, which is usually thought to have a destabilizing influence in a control system, can be used to improve the performance of a motor control system.

In the Figure 1(b) model, force feedback is implemented by feeding back a signal related to T_c. Specifically, the force-related signal contributing to e is assumed to be proportional to the mathematical integral of T_c ($F = K_F/s$ in the model block diagram) so that force feedback primarily influences postural behavior at lower frequencies. The transfer function of this model is given by:

$$\frac{BS(s)}{FS(s)} = \frac{W_{\text{prop}} \cdot NC \cdot B \cdot TD}{1 - F \cdot NC \cdot TD + W_{\text{prop}} \cdot NC \cdot B \cdot TD}. \tag{4}$$

The fit of this model, using a PD neural controller, is shown in Figure 2(c). This fit shows a gain decline and phase advance at lower frequencies that is quite consistent with the experimental data. Although others have shown that positive force feedback contributes to motor control [11], our assumption that the postural control system makes use of an integrated force-related sensory signal is based entirely on the fact that the experimental data is explained well when this form of force feedback is included in the model.

The Figure 2(c) fit now provides a good explanation of the low- and mid-frequency experimental data. However, the high-frequency data, particularly the phase data, are not well fit by this model or any of the previous models. To account for this high-frequency behavior, the Figure 1(b) model also includes a "passive" component P that contributes corrective torque T_p to the overall corrective torque T_c. T_p is assumed to arise from the stiffness and damping properties of the muscles and tendons and is proportional to BF and BF velocity ($P = K_{\text{pas}} + B_{\text{pas}}s$ in the model block diagram). There is no time delay associated with this passive torque component.

The transfer function of this model that includes both force feedback and passive torque components is given by:

$$\frac{BS(s)}{FS(s)} = \frac{P \cdot B + W_{\text{prop}} \cdot NC \cdot B \cdot TD}{1 - F \cdot NC \cdot TD + P \cdot B + W_{\text{prop}} \cdot NC \cdot B \cdot TD}. \tag{5}$$

The fit of this model is shown in Figure 2(d). This fit to the high-frequency data is now improved relative to the previous models.

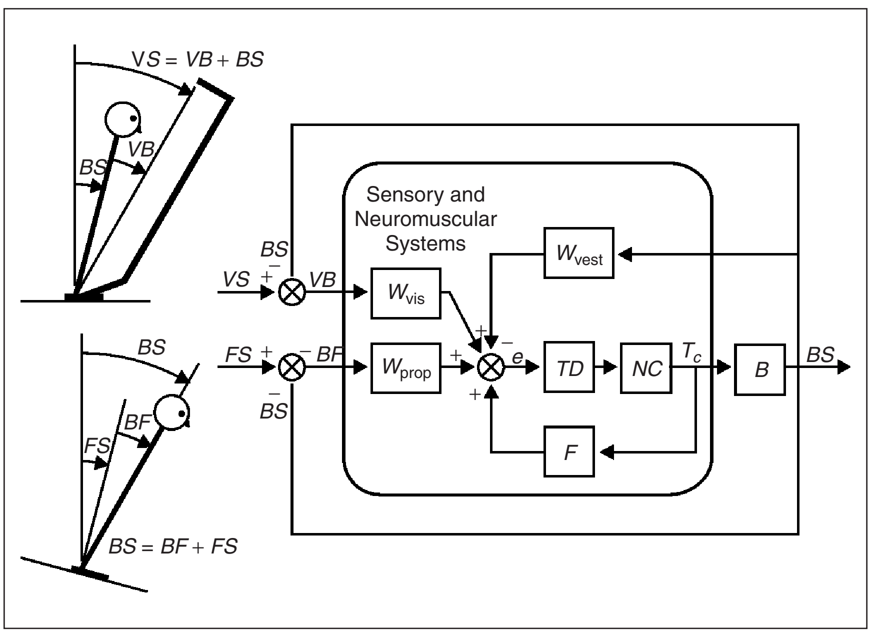

Fig. 3. A postural control model extended to include feedback from visual and vestibular sensory systems in addition to proprioceptive and force-related sensory systems shown in Figure 1. The visual system senses visual surround orientation relative to the body *VB*. The vestibular system senses body-in-space orientation *BS*. An internal orientation error signal *e* is formed from force-related feedback *F* and a weighted combination of visual, vestibular, and proprioceptive information with weighting factors W_{vis}, W_{vest}, *and* W_{prop}, respectively.

Adding Additional Sensory Information

The models shown in Figure 1 apply to a rather limited subset of the overall population (vestibular loss subjects standing with eyes closed). We now seek a parsimonious expansion of this model that includes both vestibular and visual sensory orientation cues. The model shown in Figure 3 accomplishes this by adding separate negative feedback loops for the vestibular system, which senses body orientation relative to earth-vertical (BS), and the visual system, which senses body orientation relative to the visual world ($VB = VS–BS$). "Sensory integration" is accomplished by weighted linear addition of the individual sensory channels to produce the internal error signal e. One could imagine that the nervous system selects the weights to accomplish specific tasks in different circumstances. For example, a large vestibular channel weight favors orientation to earth-vertical, while a large visual channel weight favors orientation to the visual world. However, weight changes must be performed in a coordinated fashion since changing the internal gains of a feedback control system can have a profound effect on the dynamics of the system and on overall stability.

Is there any way to estimate these sensory channel weights? As an example, consider the situation where body sway is evoked by a visual stimulus generated by tilting a visual surround about an axis through the ankle joints while the subject stands on a stationary, level support surface. In this case, sensory orientation cues are provided by visual, proprioceptive, vestibular, and force sensors. Ignoring the relatively small contribution of passive torque, the transfer function relating *BS* to *VS* is:

$$\frac{BS(s)}{FS(s)} = \frac{W_{\text{vis}} \cdot NC \cdot B \cdot TD}{1 - F \cdot NC \cdot TD + \left(W_{\text{vis}} + W_{\text{prop}} + W_{\text{vest}}\right) NC \cdot B \cdot TD}. \tag{6}$$

Note that the sensory channel weight W_{vis} occurs as a gain factor in the numerator, and the sum $W_{\text{vis}} + W_{\text{prop}} + W_{\text{vest}}$ multiplies one factor in the denominator. If we assume that the sensory channel weights represent the *relative* contribution of the different sensory channels to overall torque production, then we can assume that $W_{\text{vis}} + W_{\text{prop}} + W_{\text{vest}} = 1$. This assumption appears to be justified since experimental results show that the dynamics of the postural control system (other than the overall gain related to the value of W_{vis}) do not change in test conditions where the relative contributions of W_{vis}, W_{prop}, and W_{vest} apparently do change [6]. Transfer function data (Figure 4) obtained in experiments that used five different visual stimulus amplitudes show a family of similarly shaped transfer functions with the main difference between them being the overall gain. Coherence functions (not shown) associated with these transfer functions were similar to one another, indicating that the large gain decrease observed with increasing stimulus amplitude was not due to some saturating nonlinearity. Rather, the model-based interpretation is that the nervous system used a different combination of sensory channel weights for each of the stimulus conditions such that W_{vis} decreased and $W_{\text{prop}} + W_{\text{vest}}$ increased with increasing visual stimulus amplitudes. Functionally, this shift toward increased utilization of vestibular and/or proprioceptive sensory information reduced the postural disturbance caused by the increasing amplitude of visual surround motion.

Curve fits of (6) to the transfer functions in Figure 4 provided estimates of W_{vis} as a function of stimulus amplitude for subjects with normal sensory function (Figure 5). Also shown in Figure 5 are W_{vis} estimates for two vestibular loss subjects. Unlike the normal subjects, these vestibular loss subjects showed little change in W_{vis} with changing stimulus amplitude. Functionally, this unchanging reliance on visual cues resulted in an increasing postural disturbance with increasing stimulus amplitude such that these vestibular loss subjects had difficulty maintaining stance at larger stimulus amplitudes.

Conclusions

The models developed here should not be considered to be a definitive explanation of the postural control system. Rather, these models serve as quantitative hypotheses to help guide future research and assist with the interpretation of experi-

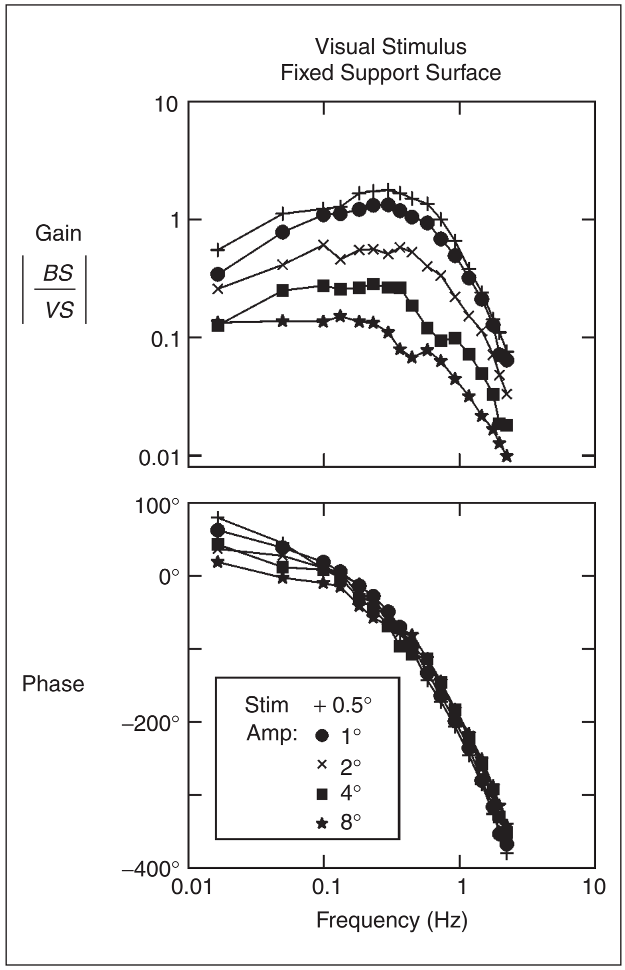

Fig. 4. Experimental transfer functions (mean of eight subjects with normal sensory function) obtained in response to pseudorandom rotation of a visual surround about an axis aligned with the ankle joint axis [6]. Each transfer function was obtained using a different stimulus amplitude (0.5-8° peak-to-peak).

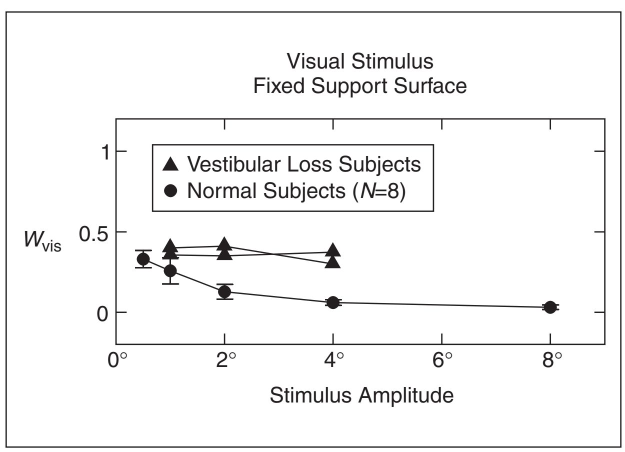

Fig. 5. Estimates of the visual contribution to postural control (visual channel weight W_{vis}) provided by curve fits of (6) to the transfer function data in Figure 4. W_{vis} is plotted as a function of stimulus amplitude for eight normal subjects (mean ±SD) and two vestibular loss subjects.

An important feature clearly revealed by the model-based interpretation of experimental data is the ability of the human postural control system to alter its source of sensory orientation cues as environmental conditions change.

mental data. For example, a control model with PD control and a positive force feedback loop [Figure 1(b)] provides a better explanation of the low-frequency dynamic behavior [Figure 2(c)] than the PID control model [Figures 1(a) and 2(b)]. Since both models have the same number of parameters, Occam's Razor favors the positive force feedback model over the PID model or any variation on the PID model that includes additional parameters. While there is some experimental evidence that positive force feedback plays a role in some aspects of motor control [11], its contribution to postural control is unknown. Our model that includes positive force feedback represents a quantitative hypothesis that motivates additional experiments to confirm or refute the contribution of positive force feedback to human postural control and to investigate the dynamic properties of this feedback loop.

An important feature clearly revealed by the model-based interpretation of experimental data is the ability of the human postural control system to alter its source of sensory orientation cues as environmental conditions change. Our relatively simple models allowed us to apply systems identification methods in order to estimate the relative contributions (sensory weights) of various sensory orientation cues in different environmental conditions [6]. However, our simple models do not predict how the sensory weights should change as a function of environmental conditions or provide insight into the neural mechanisms that cause these changes. More sophisticated models, such as those employing optimal estimation ideas [12], are needed to explore these concepts.

Acknowledgments

This work was supported by NIH grant AG17960.

Robert J. Peterka received a Ph.D. (1980) in bio-electrical engineering from Carnegie Mellon University. He is an associate scientist and head of the Human Spatial Orientation Laboratory at the Neurological Sciences Institute, Oregon Health and Science University, Portland, Oregon. His interests include application of system identification methods to physiological systems, understanding the vestibular system's contribution to eye movement and balance control, sensor fusion in sensorimotor systems, and development of improved clinical tests of vestibular and balance function.

Address for Correspondence: Robert J. Peterka, Neurological Sciences Institute, Oregon Health & Science University, 505 NW 185th Avenue, Beaverton, OR 97006 USA. Tel.: +1 503 418 2616. Fax: +1 503 418 2501. E-mail: peterkar@ohsu.edu.

References

[1] P. Gibbs and H. Sugihara, "What is Occam's Razor?," *Usenet Physics FAQ*, 1996 [Online]. Available http://www.weburbia.com/physics/occam.html

[2] S.H. Koozekanani, C.W. Stockwell, R.B. McGhee, and F. Firoozmand, "On the role of dynamic models in quantitative posturography," *IEEE Trans. Biomed. Eng.*, vol. BME-27, no. 10, pp. 605-609, 1980.

[3] R. Johansson and M. Magnusson, "Human postural dynamics," *Biomed. Eng.*, vol. 18, pp. 413-37, 1991.

[4] P.G. Morasso, L. Baratto, R. Capra, and G. Spada, "Internal models in the control of posture," *Neural Networks*, vol. 12, pp. 1173-80, 1999.

[5] R. Johansson, M. Magnusson, and M. Akesson, "Identification of human postural dynamics," *IEEE Trans. Biomed. Eng.*, vol. 35, pp. 858-869, 1988.

[6] R.J. Peterka, "Sensorimotor integration in human postural control," *J. Neurophysiology*, vol. 88, pp. 1097-1118, 2002.

[7] J.S. Bendat and A.G. Piersol, *Random Data: Analysis and Measurement Procedures*, 3rd ed. New York: Wiley, 2000.

[8] D.A. Winter, *Biomechanics and Motor Control of Human Movement*. New York: Wiley, 1990.

[9] M. Magnusson, H. Enbom, R. Johansson, and I. Pyykkö, "Significance of pressor input from the human feet in anterior-posterior posture control," *Acta Oto-laryngologica (Stockholm)*, vol. 110, pp. 182-188, 1990.

[10] V. Dietz, A. Gollhofer, M. Kleiber, and M. Trippel, "Regulation of bipedal stance: dependency on 'load' receptors," *Exp. Brain Res.*, vol. 89, pp. 229-31, 1992.

[11] A. Prochazka, D. Gillard, and D. J. Bennett, "Positive force feedback control of muscles," *J. Neurophysiol.*, vol. 77, pp. 3226-36, 1997.

[12] H. van der Kooij, R. Jacobs, B. Koopman, and F. van der Helm, "An adaptive model of sensory integration in a dynamic environment applied to human stance control," *Biol. Cybern.*, vol. 84, pp. 103-15, 2001.